

ON A CASE OF MULTIPLE NEURITIS AND CEREBRO-SPINAL MENINGITIS.¹

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THE occurrence of multiple neuritis in connection with acute and chronic infectious diseases, has for some time attracted attention, and it seems certain that there is a causative relation between the two; it has been a matter of considerable doubt whether the neuritic process is caused directly by the microbe, or whether it is rather a result of the presence in the circulation, of ptomaines or other poisons produced by bacteria in their growth. The latter view has in its favor the fact that several forms of neuritis are certainly produced by chemical poisons, and that staining and culture tests have as yet given negative results in the few cases of multiple neuritis which been examined in this way. This is, however, by no means conclusive on the point at issue, and in the present fragmentary state of our knowledge of the different kinds of bacteria the appearance under the microscope of the affected tissues may afford considerable information as to the nature of the process.

The following case may in this connection not be without interest.

Mrs. Rolf; age 27. The patient was taken rather suddenly a year ago with violent pains in the arms and legs, followed by paralysis and marked atrophy in all four extremities. She was at that time helpless and unable to feed herself. She was not unconscious, and there seem to have been no cerebral symptoms. The knee jerk is said to have been retained. She recovered from this illness very gradually, and regained considerable power in her arms and legs, so that she was able to be about.

Two weeks ago the patient was taken ill in the same way as before, with violent pains and great prostration,

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some fever. Three or four days ago she passed into a condition of stupor, which has persisted since.

On examination the patient makes unintelligible answers to questions, puts out her tongue feebly when asked to do so, is very quiet unless disturbed. When moved she cries out as if in pain; the neck and all four extremities are tender to pressure. There is opisthotonos which is not very marked. The patient is markedly emaciated, and the muscles of all four extremities are considerably atrophied, especially the thenar muscles and interossei. There seems, however, to be no decided paralysis, as the patient moves both arms and legs occasionally.

There is no facial paralysis; the pupils are equal and react to light; the fundus oculi shows no pathological change, although the optic discs are very pink; the triceps and knee reflexes are quite marked, in fact seem somewhat exaggerated. Electric examination with the faradic current attempted, but unsatisfactory on account of the extreme tenderness of the extremities.

No specific history is obtainable. The patient has never been an habitual drinker.

Provisional diagnosis. Tubercular meningitis of the cord, multiple neuritis a year ago. Examination of the urine for lead recommended.

The stupor continued for two days, when the patient died.

Autopsy twelve hours after death. Lungs congested and œdematous. Two encapsulated masses on right side of vertebral column, adherent to diaphragm, consisting of calcareous matter. Kidneys and liver appear normal.

Brain, dura mater normal, pia much congested over the vertex, with marked serous effusion. Brain substance throughout appears normal. Medulla very firm, otherwise of normal appearance.

Dorsal and lumbar portions of cord and a portion of the ulnar nerve were removed and examined. Pia mater of cord much congested, the veins very tortuous, with serous effusion which has accumulated about the lower part of the cord. On section of the cord and ulnar nerve nothing abnormal was to be seen.

The tissues were hardened in Müller's fluid, and stained with chloride of gold, alum carmine, hæmatoxylin and rosin, and by the Weigert method.

Microscopical examination of the brain, medulla and cerebellum reveals nothing abnormal.

In the ulnar nerve there is increase of the connective tissue elements in almost all parts of the section, and namely a thickening of the septa between the fasciculi, and a loose network between these and the nerve fibres, with interstices which look as if they might have been filled with serum; there is also an increased number of nuclei in the sheaths of Schwann; there are no collections of leucocytes, and nothing to indicate an acute purulent or tubercular process. Carmine and hæmatoxylin staining shows a moderate number of swollen axis cylinders. The myeline sheaths appear many of them swollen and granular.

In the spinal cord there is no thickening of the meninges, the white matter of the cord appears normal throughout, as does also the network of fibres in the gray matter. In the lumbar and sacral portions the nerve-cells of the anterior horns of gray matter are considerably shrunken away from the surrounding tissue, most of them appear to have neither axis cylinder nor protoplasmic processes, in many of them the nuclei are pushed to one side of the cell, so as to form a distinct projection upon the outline of the cell-body; in a few of the cells there are vacuoles, occupying a greater or less part of the protoplasm of the cell. In some of the sections there are in the anterior horns of gray matter, numbers of small bodies of about the size of the so-called corpora mylacia; they have however a more granular appearance than the latter; whether they are portions of degenerated ganglion cells I am unable to say.

In the anterior and posterior nerve roots the myeline sheaths appear many of them swollen and granular, there seems to be little if any increase of the connective tissue.

In the dorsal portion of the cord the anterior horns of gray matter and columns of Clarke appear normal, as does the white matter throughout.

In interpreting the changes described above, there is every probability from the appearances on naked eye examination at the autopsy that the case was one of cerebrospinal meningitis of the infectious variety; this disease is endemic in Cleveland, and two similar cases chanced to come under my observation within a week of the time at which this case occurred, one of which was verified by post-mortem examination.

The changes in the ulnar nerve indicate an overgrowth of connective tissue, which probably dates from the attack of the preceding year, together with an acute inflammatory condition, evidenced by the swollen axis cylinders, broken down myeline sheaths, and possible accumulation of serum within the neurilemma.

The changes in the nerve-cells are such as have been often described, and doubt has been cast on their significance. That the changes in this case are to some degree pathological, seems to me to be proved by the occurrence of vacuoles, and the pushing to one side of the nuclei; the unusual shrinkage of the cells during hardening, and loss of the cell processes are also significant. Whether these changes are primary, or simply a result of the preceding attack of multiple neuritis, it is hardly possible to decide; the probability is that they are secondary.

No examination of the tissues for bacteria was made.

In reviewing the history of the case in the light of the pathological changes which were found, it seems clear that multiple neuritis was present from the outset; the friends of the patient were positive that on the occasion of the first attack there were from the beginning severe pains and tenderness in the limbs, with little if any stupor or loss of mental power; so that we may conclude that the inflammation of the nerve-trunks was coincident with, if it did not precede, the meningitis.

The appearances in the ulnar nerve do not correspond with those described as occurring either in ordinary Wallerian degeneration, or in the usual forms of multiple neuritis; aside from the thickening of the neurilemma, the changes would all be very readily explained by supposing

congestion and a serous exudate of sufficient severity to cause pressure on, and consequent changes in some of the nerve tubes; the membranes of the nerve trunks, usually called the neurilemma, seem to have been primarily affected, and this with the very early appearance of the neuritis tends strongly to show that the inflammation was directly infectious in origin, rather than an indirect result of the meningitis.

The occurrence of multiple neuritis with cerebro-spinal meningitis was first suggested by Mills, of Philadelphia, in 1888, although unfortunately in his cases he could obtain no autopsy. He found in the acute stage, as in this case, symptoms of an irritative rather than of paralytic lesion. This is readily understood if we suppose the process to be identical in nature with that in the pia mater of the cord and brain.

In the epidemics of multiple neuritis which have been described, it is probable, as has been pointed out by Eisenlohr, that the specific cause is the same as that for cerebro-spinal meningitis, but definite knowledge on this point can only be furnished by further investigation, especially in the line of bacteriological research.